As already pointed out, the majority of bacilli carriers who have been the cause of typhoid fever in others, have been persons who handled certain food products, especially those used in a raw state. This suggests a practicable way of dealing with a source of infection of this kind. When a bacilli carrier is discovered among the employees of a dairy, restaurant, or other food-producing or food-distributing establishment, such an employee can be compelled, under the new health laws of Indiana, to seek other employment. The danger of his spreading infection will thus be greatly reduced, although not entirely eliminated.

In conclusion, and by way of summary, I may say, first, there is ample evidence for considering the danger of the spread of typhoid fever by chronic bacilli carriers as worthy of the attention of health officers; second, epidemics caused by bacilli carriers are characterized by the occurrence of small groups of cases at intervals of a few months in the immediate vicinity of the carrier, or in the territory supplied by some food product handled by him; third, the most practicable way of reducing the danger to the public from these individuals is by excluding them from work that brings them in contact with food products.

CHRONIC NEPHRITIDES, DUE TO ACUTE SYSTEMIC INFECTION.

By Heinrich Stern, M.D.,
PHYSICIAN TO ST. MARK'S HOSPITAL, NEW YORK.

In spite of the universally accepted fact that chronic renal disease may be one of the more or less remote sequences of a systemic infection, it is, nevertheless, true that entirely too little attention has been and is bestowed upon the connection between acute systemic infection, that is, the transmission of the infective agent by the blood current, and a considerable number of permanently injured kidneys. The infectious origin of a chronic nephritic state is generally only then recognized when it has developed as the direct continuation of an attack of acute or subacute nephritis that has arisen on the basis of an acute infection, or when the chronic nephritis has supervened in the course of long-established or chronic instances of infections like malaria, tuberculosis, or syphilis.

It has ever been the dictum of experienced clinicians that urinary findings like albumin, large numbers of blood cells and renal epithelium, and epithelial and granular casts can be attributed to a systemic infection of the kidney only when originating in the course of a pronounced acute infectious disease. While this clinical viewpoint leaves nothing to be desired as far as conservatism is concerned, the physician generally cannot furnish any other but clinical evidence that in a given case the renal involvement is due to a systemic invasion. To prove positively a causal connection in such instances is extremely difficult or well nigh impossible when the patient remains alive, and the necropsy may or may not reveal the specific pathogenic bacteria in the kidneys. Still more difficult it would be to establish an etiological relationship between an existing chronic nephritis and a systemic infection that had been of a more or less transitory nature.

In cases of this kind the effect endures while the cause has been removed months or even years ago, and it is, of course, out of the question to be able to demonstrate the original materia peccans in either blood or kidneys. Nevertheless, the great amount of accumulated clinical evidence prompts us to regard acute systemic infection as one of the causes of chronic renal disease.

The scope of this article is limited to a consideration of the relationship of acute systemic infection to chronic nephritides; it embraces, therefore, clinical experiences concerning acute infectious diseases, like scarlet fever, diphtheria, pneumonia, influenza, measles, etc., which may give occasion to chronic renal damage, but does not include any data anent renal involvement as the consequence of gonorrhea and affections starting from the lower genito-urinary tract, which may invade the kidneys in a direct manner, and chronic infections like tuberculosis, syphilis, or chronic forms of malaria.

The typical renal involvement concomitant with or following an acute infectious disease is of a parenchymatous nature. The chronic nephritic states evolving from the acute processes, it stands, therefore, to reason, tend to assume or rather to perpetuate this parenchymatous character. Roughly speaking, however, not more than 50 per cent. of the pertaining chronic nephritides are predominatingly parenchymatous. In about 20 per cent. of the cases indurative processes follow in the wake of acute parenchymatous inflammation, or supervene in the presence of subchronic or chronic parenchymatous states, and in at least 30 per cent. of the cases amyloid lesions concur with the original parenchymatous affection.

The lesions in some forms of acute nephritis are of brief duration, and the kidneys recover their normal state and function just as do the lungs after an attack of lobar pneumonia. Other types of acute nephritis are characterized by lesions tending to chronicity, and the original condition of the kidneys will never be restored. In nephritides ensuing in or following scarlatina and diphtheria, some of the lesions are usually of a permanent nature; the same is also the case in nephritic states due to other acute infectious diseases, although the permanent lesions occur here much less frequently and are generally of a milder degree. With these irreparable lesions as a foundation, true nephritis of a chronic nature will sooner or later supervene in nearly every case.

The direct transformation of an acute nephritis into a chronic form has been observed in comparatively few instances only; however, the successive transition from an acute nephritic process through subacute and subchronic stages, to the chronic condition can be traced clinically with more or less accuracy in a considerable number of cases. The successive transformation may occur within some weeks or may take many years. When the process is diffuse and purely parenchymatous, it has the disposition rapidly to assume the chronic form.

There are cases in which a chronic nephritis is not recognizable for long periods, in which there is no demonstrable connection between an acute renal inflammation which has occurred many years previously and an existing chronic nephritis. In such instances we may have to deal with indurative processes of varying degree and intensity. Renal induration may either set in when the parenchymatous nephritis is still fresh, or it may not be called forth until it has progressed to a certain stage. It is also possible that latent or limited indurative metamorphosis is occasionally present prior to the establishment of the parenchymatous inflammation, and that the latter stands at the foundation of a renewed or greater activity of the process of induration. Preëxisting indurative lesions, however, bear a primary and not a secondary character, as do the particular indurative occurrences under consideration.

Secondary renal induration, which finally leads to sclerotic kidney, may form a terminal stage of either acute, subacute, or subchronic conditions of parenchymatous inflammation. After a time, the indurative process which has brought the parenchymatous degeneration to a standstill, may itself attain a certain degree of latency. It is during this period of comparative latency, which may occasionally endure for a number of years, that the existing renal lesions remain often unrecognized, and that the connecting traces between the original acute renal inflammation and an eventual conspicuous final nephritis become more or less obliterated. On account of the concealed syndrome during the latent period of induration, the final nephritis which evinces itself not only by the urinary findings, but also by well-authenticated systemic phenomena, is often erroneously held to be a comparatively new affection. This is especially the case when the patient has just passed through an attack of illness, which may have stimulated the old nephritic process to renewed activity. At other times, the manifest nephritic state has been described, especially by the patient himself, as being of spontaneous production.

An acute infectious disease may thus be the original cause of a chronic renal affection in which indurative processes predominate. Progressive renal induration occurs so frequently in the absence of any determining systemic factor and in individuals who had at one time been affected with an acute infectious nephritis, that I have to reiterate my opinion, expressed on a former occasion,[1] namely, that the majority of chronic interstitial nephritides, not due to constitutional causes, is the consequence of an acute infectious disease.

The affected kidneys become readily subject to amyloid degeneration. This may ensue on top of the diffuse (parenchymatous) processes within a few months after the onset of the latter. The amyloid transformation, however, which interests us most on this occasion is that which occurs together with chronic parenchymatous or secondary indurative nephritis. Amyloid degeneration of the kidneys, always supervening in both organs, is never a primary process. It is nearly always produced in the wake of certain cachectic conditions, the underlying abnormal blood composition of which may have been evoked by long-continued renal disease. Not infrequently amyloid kidney is mistaken for parenchymatous or indurative nephritis. With the former it has, in common, the large amount of urinary albumin, with the latter the scanty number of morphotic elements in the urine; these consist in the greater part of hyaline and finely granular casts. In the rarest of instances amyloid degeneration is traced back to an acute nephritic attack.

The history of an individual with chronic nephritis will often reveal the fact that he has at one time been afflicted with an acute nephritis, following an attack of an infectious disease like scarlet fever, diphtheria, pneumonia, influenza, measles, erysipelas, rheumatic or typhoid fever, smallpox, parotitis, etc., and that soon afterward the nephritic symptoms have vanished. In many of the pertaining cases, however, there ensued but a temporary arrest of the nephritis, and in the absence of subsequent urinary examinations, this temporary cessation was translated to mean a permanent amelioration. Furthermore, the abatement of the albuminuria has been, and still is often, interpreted as the abatement of the nephritic process itself, a misconception of facts to which many a fatality has to be ascribed. Albuminuria may occur without any inflammatory renal changes, as I have again shown in a recent communication,[2] and in certain nephritic states the albuminuric phenomenon may not ensue at all for a long period or may be temporarily suspended.

The records of the last 200 cases of chronic nephritis that have come under my observation give the history of one or more acute infectious diseases at some period during the life of the patients in 192 instances. Among these an acute renal involvement had been positively set up in at least 121 cases. Moreover, the histories following the attack of the infectious disease in 56 or 58 of the remaining cases strongly point to the occurrence of an acute nephritic process. (Among these should be included 4 cases of tuberculosis, 5 cases of syphilis, and 1 case of chronic malaria.) Ignoring, however, all the unconfirmed instances, we find that renal involvement had existed in 62.5 per cent. of the cases that had been acutely infected. The frequency of renal involvement after the various infectious diseases was as follows: scarlet fever, 38 cases; diphtheria, 35 cases; influenza, 16 cases; pneumonia, 12 cases; measles, 12 cases; typhoid fever, 6 cases; rheumatic fever, 1 case; erysipelas, 1 case.

---

[1] Heinrich Stern, The Renal Complications and Sequelæ of Influenza, Med. Rec., January 11, 1908.

[2] Heinrich Stern, Compensatory Albuminuria, Med. Rec., June 26, 1909.

The chronic nephritic processes evolved from or supervening some time after the occurrence of the 121 positive acute nephritides exhibited a preponderating parenchymatous character in 58, the features of secondary induration in 24, and of amyloid degeneration in 39 instances.

Of the 200 quoted instances of chronic nephritis, 156 occurred in men, and 44 in women. This, however, is not the normal proportion of the sexes as affected by chronic nephritis, for the reason that my records comprise the histories of a comparatively large number of male applicants for life insurance who were referred to me after they had been rejected by the examiners of the companies. The age of the youngest individual was eight, of the oldest, sixty-four years.

The 121 cases of chronic nephritis furnishing a positive history of acute renal involvement during or after an infectious disease, had ensued in 97 men and 24 women. The ages varied between eight and forty-nine years. (None of the older individuals could produce definite data concerning the acute nephritis in the wake of an infectious disease.) The time which had elapsed since the occurrence of the acute nephritic state amounted to from ten months to twenty-six years. As a matter of course, the indurative instances were those which had lasted longest.

From the foregoing remarks it does by no means follow that an acute nephritis of infectious origin always stands in direct relationship to a chronic nephritic state that supervenes in after years. However, we are justified in assuming that kidneys, acutely affected at one time, are thereafter possessed of a lowered degree of resistance in many, and of a diminished functional activity in some, of the instances. Exciting causes which will not readily affect the entirely sound kidney to any extent, bear upon a weakened organ and tend to call forth therein further damage. Exposure to telluric conditions of cold and dampness, continued physical and mental strain, excesses in baccho et venere, factors which are generally held to be excitants of renal disease, will be conducive to the production of nephritis in proportion to the lack of resistance of the kidneys. Even the slightest provocation may affect the non-resistant kidney and occasion intermittent or permanent manifestations of an active morbid process. While, therefore, an acute nephritis is not necessarily followed by a chronic degenerative renal state directly traceable to it, it evidently prepares the soil for subsequent invasions and textural and functional decline.

The following conclusions are offered:

1. Kidneys, acutely affected at one time, are thereafter possessed of a lowered degree of resistance in many, and of a diminished functional activity in some, of the instances.

2. It is very difficult, so far as positive demonstration is concerned, to establish an etiological relationship between an existing chronic nephritis and a systemic infection that had been of a more or less transitory nature. Clinical evidence, however, prompts us to regard acute systemic infection as one of the causes of chronic renal disease.

3. The typical renal involvement concomitant with or following an acute infectious disease is of a parenchymatous nature. About half of the chronic nephritides evolving from nephritic processes that have originated during or after an acute infectious disease, are predominatingly parenchymatous; in about 20 per cent. of the cases indurative processes follow in the wake of acute parenchymatous inflammation, or supervene in the presence of subchronic or chronic parenchymatous states; and in about 30 per cent. of the cases amyloid lesions concur with the original parenchymatous affection.

4. The direct transformation of an acute nephritis into a chronic form has been observed in comparatively few instances only; however, the successive transition from an acute nephritic process through subacute and subchronic stages to the chronic condition can be traced clinically with more or less accuracy in a considerable number of cases.

PERNICIOUS ANEMIA, WITH REMISSION FOR SEVENTEEN YEARS.

By Alexander McPhedran, M.D.,
PROFESSOR OF MEDICINE IN THE UNIVERSITY OF TORONTO.

The object of this article is chiefly to record a case of pernicious anemia, in which there was a remarkably long period of complete remission of symptoms, amounting to a cure for some sixteen years, with final relapse, showing all the characteristic symptoms and pursuing a truly progressive course to a fatal ending.

The case, that of a physician, was one of five previously reported.[1] At that time Dr. P., aged forty-nine years, was engaged in a large country practice. He had been losing strength and growing anemic for a year or two. He became much worse, and was compelled to cease work in the spring of 1889; he soon became very ill, with a temperature of 104° F. and marked delirium. There was severe

[1] Medical News, October 11, 1890.